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Chapter 4

Vastus lateralis surface and single motor unit EMG following submaximal shortening and lengthening contractions

Abstract

It is well known that isometric force is depressed immediately following shortening and enhanced following lengthening. Thus the force generating capacity of muscle fibres changes during a single shortening or lengthening contraction. Therefore, we hypothesize that when the same submaximal torque had to be generated immediately following shortening, muscle activation (recruitment and discharge rate of motor units) had to be increased, while a lower activation would suffice to produce the same torque following lengthening. Knee extensor muscle activation following shortening and lengthening (20° at $10^\circ \cdot s^{-1}$) was determined using rectified surface electromyography (rsEMG) a first session (10 and 50 % MVC) and additionally with EMG of 42 vastus lateralis (VL) motor units recorded during a second session (4 – 47 % MVC). RsEMG and discharge rates following shortening and lengthening were normalized to isometric reference contractions. As expected, and independent of torque level normalized rsEMG (1.15 ± 0.19) and discharge rate (1.11 ± 0.09) were higher 1 s following shortening and gradually declined during the next 4 s of isometric torque production ($P < 0.05$). One second following lengthening normalized rsEMG (0.91 ± 0.10) was, as expected, lower than 1.0 ($P < 0.05$), but normalized discharge rate (0.99 ± 0.08) was not ($P > 0.05$). Both parameters did not change during the next 4 s. Thus, muscle activation was increased to compensate for a reduced force capacity following shortening by increasing the discharge rate of the active motor units (rate coding). In contrast, following lengthening rsEMG decreased while discharge rates of active motor units remained similar, suggesting that derecruitment of units might have occurred.

Introduction

It is well known for isolated skeletal muscle (fibres) that the maximal isometric muscle force is depressed following a phase of loaded shortening and the maximal isometric muscle force is enhanced following a phase of loaded lengthening compared to isometric contractions. These phenomena are referred to as shortening induced force depression and lengthening induced force enhancement respectively (Abbott and Aubert, 1952; Edman *et al*, 1993; Edman *et al*, 1982; Granzier and Pollack, 1989; Marechal and Plaghki, 1979). More recently, force depression has been demonstrated in both maximal stimulated contractions of the adductor pollicis (de Ruiters and de Haan, 2003; de Ruiters *et al*, 1998; Lee and Herzog, 2003) and maximal voluntary contractions of the adductor pollicis and the quadriceps muscle (de Ruiters and de Haan, 2003; Lee and Herzog, 2003; Lee *et al*, 1999; Lee *et al*, 2000). From these studies it is known that force depression increases with increasing shortening amplitude and with decreasing speed (increasing force) of shortening (Abbott and Aubert, 1952; de Ruiters and de Haan, 2003; de Ruiters *et al*, 1998; Lee and Herzog, 2003; Marechal and Plaghki, 1979). Moreover, force depression seems to be directly related to the mechanical work performed (Herzog *et al*, 2000). Although frequently investigated, the mechanism underlying force depression is not completely understood. The most likely explanation for the depressed force following shortening is the theory of stress-dependent inhibition of cross-bridge attachment (Herzog, 1998; Marechal and Plaghki, 1979). Recently it was found that this inhibition is not caused by an “action distortion” as proposed by Herzog (1998), but might reside in the cross-bridges themselves (Herzog and Leonard, 2007).

The enhancement of force following lengthening has been shown in the adductor pollicis muscle during both maximal stimulated (de Ruiters *et al*, 2000; Lee and Herzog, 2002) and maximal voluntary (Lee and Herzog, 2002) contractions, but not during maximal voluntary contractions of the quadriceps muscle (Hahn *et al*, 2007). Force enhancement is known to increase with increased stretch amplitude and is independent of the speed of the lengthening (Abbott and Aubert, 1952; Cook and McDonagh, 1995; de Ruiters *et al*, 2000; Lee and Herzog, 2002). Furthermore, although force enhancement is more pronounced on the descending limb of the force – length relationship, it could also be obtained on the ascending limb (Herzog and Leonard, 2002). The mechanism underlying force enhancement is probably associated with strain building up during lengthening in viscoelastic components, potentially titin, outside the cross-bridges (de Ruiters *et al*, 2000; Edman and Tsuchiya, 1996; Herzog and Leonard, 2005; Herzog *et al*, 2003; Pinniger and Cresswell, 2007).

Although less work has been performed on submaximal contractions, both depression and enhancement of force have been demonstrated during submaximal stimulated (de Ruiter *et al*, 1998; Pinniger and Cresswell, 2007) and submaximal voluntary (Oskouei and Herzog, 2006a; Oskouei and Herzog, 2006b; Pinniger and Cresswell, 2007; Rousanoglou *et al*, 2007) contractions. Moreover, when force was controlled during submaximal contractions, isometric surface electromyography (EMG) of the adductor pollicis was found to be higher following shortening (Rousanoglou *et al*, 2007) and lower following lengthening (Oskouei and Herzog, 2005; Oskouei and Herzog, 2006b) compared to isometric contractions at the same muscle length without a preceding lengthening or shortening phase. However, it has never been investigated at the motor unit level how muscle activation (defined as the number of motor units recruited and their discharge rate) is adapted to the reduced force capacity following shortening and the enhanced force capacity following lengthening. Given the different underlying mechanisms for both phenomena it is conceivable that muscle activation is adapted differently following shortening and lengthening contractions. Moreover, muscle activation following shortening and lengthening have been studied in the small adductor pollicis muscle (Oskouei and Herzog, 2005; Oskouei and Herzog, 2006b; Rousanoglou *et al*, 2007) but never in the larger quadriceps muscle. The purpose of the present study was therefore to establish at the motor unit level whether quadriceps muscle activation was different during isometric contractions following shortening and lengthening compared to isometric reference contraction at the same joint angle (i.e. muscle length) and the same absolute isometric torque. We hypothesized that, due to the lower force capacity of muscle fibres following shortening, muscle activation would be higher following shortening. In contrast, we expected muscle activity to be lower following lengthening, due to the enhanced force capacity of the active muscle fibres. In the present study muscle activation was quantified based on the surface EMG of the quadriceps femoris muscle and the discharge behaviour of single motor units of the vastus lateralis (VL) muscle.

Methods

Subjects

Ten healthy subjects (5 men and 5 women) with a mean \pm SD age of 26.7 ± 6.1 yr, height 178.2 ± 9.0 cm and mass 70.3 ± 8.1 kg voluntarily participated in two sessions. The study was performed with the approval of the ethics committee of the VU University Medical Center in Amsterdam, The Netherlands, and in accordance with the Declaration of Helsinki. After written and verbal explanations of the objectives and

procedures of the experiments, the subjects signed informed consent. All subjects refrained from heavy exercise 48 hours prior to the experiments.

Torque recordings

Isometric and isokinetic knee extension contractions were performed on a specially designed dynamometer, which recorded the exerted torque at its axis of rotation. Subjects sat in an upright position with a hip angle of 85° (0° = full extension), straps restrained hip and shoulders. A cuff was fastened around the lower leg and was subsequently attached to the lever of the dynamometer. A shin guard ensured subjects could exert maximal forces without discomfort at the shin. The axis of rotation of the dynamometer was aligned with the lateral femoral condyle, while subjects delivered a torque of about 20 % of their maximal voluntary contraction (MVC) torque (the expected average torque for motor unit recording) at a knee angle of 60° (0° = full extension). Contraction torques were AD-converted and stored on disk for off-line analysis. All recorded torques were corrected for the effect of gravity at all knee angles.

Experimental protocol

Subjects performed isometric contractions following muscle shortening and lengthening and isometric reference contractions. Subjects were asked to track a trapezoidal torque trajectory that was displayed on the computer monitor (Figure 4.1). A slow isometric ramp up (5 s duration) was performed to a constant absolute torque, followed by a torque plateau of 10 s and a 5 s ramp down. For the shortening (Figure 4.1, left panel) and lengthening (Figure 4.1, right panels) contractions, the torque plateau consisted of a 3 s isometric contraction at the initial knee angle, followed by a 2 s shortening or lengthening of the quadriceps muscle, followed by a further isometric contraction of 5 s at the final knee angle. The torque was hold for 5 s following shortening and lengthening to monitor changes in muscle activation following movement. Shortening and lengthening were always performed at an angular velocity of $10^\circ \cdot \text{s}^{-1}$ over a trajectory of 20° (around optimum knee angle for torque production, which was on average 62.5°). The isometric reference contractions were performed at the final knee angles (52.5 and 72.5°). Torque should be kept constant during the 10 s torque plateau, even while the knee angle was changing during the dynamic part of the torque plateau (for shortening lengthening contractions). Isometric contractions and shortening and lengthening contractions were performed twice in random order. During all contractions on line torque traces were visible on a computer screen and subjects were verbally encouraged to follow the trapezoidal traces as closely as possible. At the end

of the first and second session, subjects were asked to perform flexion MVC at the optimum knee angle with surface EMG of the biceps femoris (BF) and semitendinosus (ST) recorded for normalization of the hamstring surface EMG during the sub maximal contractions.

In the first session the potential effects of force depression and enhancement on muscle activation were studied using surface EMG during constant torque contractions performed at 10 and 50 % of the MVC torque produced at optimum knee angle. In the second session the potential effects of force depression and enhancement on muscle activation were studied using both surface and intramuscular wire EMG during contractions at which the torque was chosen approximately 20 % above the recruitment threshold of the identified motor units (range 4 – 47 % MVC). In an additional session prior to the two experimental sessions, subjects practiced tracking the trapezoidal torque trajectory.

Surface electromyography

Surface EMG data of 5 muscles, VL, vastus medialis (VM), rectus femoris (RF), BF and ST, was obtained during the first session. Electrical activity of the BF and ST were recorded to determine co-contraction. For the VL muscle two electrode pairs were placed on the muscle. The first pair was placed on the part of the muscle from which in the second session motor unit EMG would be obtained (referred to as mid VL position). The second electrode pair was placed more distally on the VL muscle (referred to as distal VL position). This was done to determine if possible effects of knee angle and contraction mode on surface EMG were similar in the mid VL position compared to the more distal VL position the surface EMG is usually recorded from. During the second session surface EMG was only obtained from the mid position of the VL muscle, the electrodes were placed on both sides of the intramuscular wire electrodes in that session (see Intramuscular electromyography). Furthermore, during the second session only surface EMG from the BF was obtained to monitor co-contraction.

EMG was recorded using surface electrodes (Blue Sensor, Ambu, Ølstykke, Denmark, lead-off area: 1.0 cm²). After shaving and cleaning the skin with 70 % ethanol two electrodes were placed on the belly of each muscle in a bipolar configuration, in line with the muscle fibre direction and with an inter-electrode distance of 25 mm (centre to centre). For the mid VL position the inter-electrode distance was 5 mm greater (30 mm), as they were placed on both sides of the intramuscular wire electrodes in the second session. Reference electrodes were placed on the right patella and on the lateral epicondyle of the femur of the right leg. The location of each

electrode was accurately marked with a waterproof felt tip pen for precise electrode re-application in the subsequent session. Surface EMG signals were amplified (x1000) with a biosignal amplifier (g.tec, Austria, 10-500 Hz, input impedance 110 MOhm), AD-converted with a Simultaneous-Sampling AtoD board (PCI-6143, National Instruments, America), digitized (10 kHz), band-pass filtered (4th order Butterworth, bi-directional, 10-400 Hz) and stored with the torque signal on computer disk.

In pilot experiments twitch stimulation was applied to the femoral nerve and the M-wave shapes were studied to confirm that the EMG electrodes remained distal to the motor end plate zone following a change in knee angle: M-wave amplitude was found to be constant for the range of knee angles used in the present study and there was no change in the order of occurrence of the positive and negative phase of the M-wave.

Intramuscular electromyography

The method to record and analyze single motor unit action potentials (SMUAPs) has been described in more detail before (de Ruiter *et al*, 2005; de Ruiter *et al*, 2004). Custom-made, fine-wire electrodes were constructed from four polytereftalate-butylene-coated wires (0.044 mm diameter, stainless steel core, Capable, The Netherlands) to record SMUAPs. The four wires with a length of 10 cm were glued together at the tip and cut transversely, passed through a hypodermic needle (0.75 x 40 mm) and bent backwards over the needle tip for the last 2 mm. The needles including the electrodes were sterilized for 30 min at 150° C. A new needle-electrode was used for each measurement. Before insertion, the skin at the electrode site was shaved and cleaned with 70 % ethanol. The needle-electrode-combination was inserted 40 mm deep in the middle part of the belly of the vastus lateralis muscle and the needle subsequently withdrawn, leaving the quadrifillar tip of the electrode in the muscle. After insertion the subjects were asked to perform a few maximal (brisk) voluntary contractions to stabilize the position of the electrode in the muscle. Six bipolar EMG signals were led off from the four free ends of the electrode which were connected to 50-cm-long isolated cables. The signals were amplified (x1000) with a biosignal amplifier (g.tec, Austria, 0.01-10 kHz, input impedance 110 MOhm), AD-converted with a Simultaneous-Sampling AtoD board (PCI-6143, National Instruments, America), digitized (44.1 kHz), played through loud-speakers online and saved on computer disk. A reference electrode was placed on the patella of the right knee. Following band-pass filtering (1-10 kHz) the six bipolar signals were displayed immediately on-screen.

Intramuscular EMG was considered stable and selective, when one or two

motor units had a distinctive amplitude and shape on at least one of the six EMG channels during a few submaximal isometric contractions. If there was no unit which could be selectively recorded after insertion of the wire electrode, the electrode was slightly re-positioned by gentle pulling until selective recording became possible. The discharge behaviour of the same motor units was studied following shortening and lengthening contractions and during isometric reference contractions.

EMG analysis

Rectified surface EMG amplitude (rsEMG) and motor unit discharge rates, the inverse of the inter-SMUAP intervals, were calculated 1 to 5 s (1 s intervals) following shortening and lengthening and at the same time points during the isometric reference contractions (Figure 4.1). RsEMG and discharge rates of the two attempts of each contraction were averaged. To correct for differences in absolute rsEMG and discharge rates among subjects, muscles and torque levels, isometric rsEMG and discharge rates following shortening and lengthening were normalized to the isometric reference rsEMG and discharge rates at the same knee angle. When normalized rsEMG and motor unit discharge rates following shortening were enhanced (> 1.0), this was taken as an indication that force depression was present and compensated for by increased muscle activation. When normalized rsEMG and motor unit discharge rates after lengthening were lower than 1.0, this was taken as an indication for force enhancement. Antagonist (ST and BF) rsEMG was normalized to their maximum values obtained during flexion MVC.

For the intramuscular EMG data of the second session, SMUAPs were identified on the basis of their amplitude and shape using custom-written Matlab software (de Ruiter *et al*, 2005; de Ruiter *et al*, 2004). Usually, small changes in SMUAP amplitude and/or shape occurred gradually during the different contractions (Figure 4.1), due to small and unavoidable changes in position. Therefore, in the analysis, motor unit templates were made and updated within and also between contractions. The discharge behaviour during shortening and lengthening will not be presented in the present manuscript.

Statistical analysis

Data are presented as mean \pm SD. For the first session for all subjects the mean values of the different quadriceps muscles of the absolute isometric rsEMG following shortening and lengthening and the absolute isometric rsEMG during isometric reference contractions at both torque levels (10 and 50 % MVC) and at the different

time points were used for statistical analysis using repeated measures ANOVA. Subsequently, to test for differences in time and between torque levels, ANOVA for repeated measures was performed on the normalized rsEMG following shortening and lengthening. For rsEMG and discharge rates of each subject in the second session, paired samples t-tests were used to determine whether the normalized rsEMG and discharge rates 1 s following shortening or lengthening were significantly different from 1.0 (isometric reference contraction at the same knee angle). ANOVA for repeated measures was used to test for changes during the 5 s sampling period following shortening and lengthening. For each subject the averaged value of the normalized discharge rates of different motor units was used in the statistical analysis ($n = 10$).

Furthermore, ANOVA for repeated measures was performed on the normalized BF and ST rsEMG following shortening and lengthening to test for differences in co-contraction in comparison with the isometric reference contractions. If significant main effects were observed Bonferroni tests were performed for post hoc analysis. The level of significance of all statistical analyses was set at $P < 0.05$.

Results

Verification of the data

Average torque values 1 – 5 s following shortening and during isometric contractions at the same knee angle respectively were 28.3 ± 6.9 and 28.0 ± 7.0 Nm at 10 % MVC (first session, $P > 0.05$), 139.4 ± 34.4 and 139.7 ± 34.6 Nm at 50 % MVC (first session, $P > 0.05$) and 53.8 ± 22.4 and 53.5 ± 22.4 Nm at 18.1 ± 13.3 % MVC (second session, $P > 0.05$). Average torque levels 1 – 5 s following lengthening and during isometric contractions at the same knee angle respectively were 27.8 ± 6.6 and 28.0 ± 6.9 Nm at 10 % MVC (first session, $P > 0.05$), 139.4 ± 34.5 and 139.4 ± 34.4 Nm at 50 % MVC (first session, $P > 0.05$) and 53.1 ± 22.6 and 53.3 ± 22.7 Nm at 18.7 ± 13.3 % MVC (second session, $P > 0.05$). Thus, during the whole sampling period, there were no differences in the absolute torque between contractions following shortening and lengthening and isometric contractions, indicating that any potential differences in muscle activation between these contractions will not be caused by differences in torque levels.

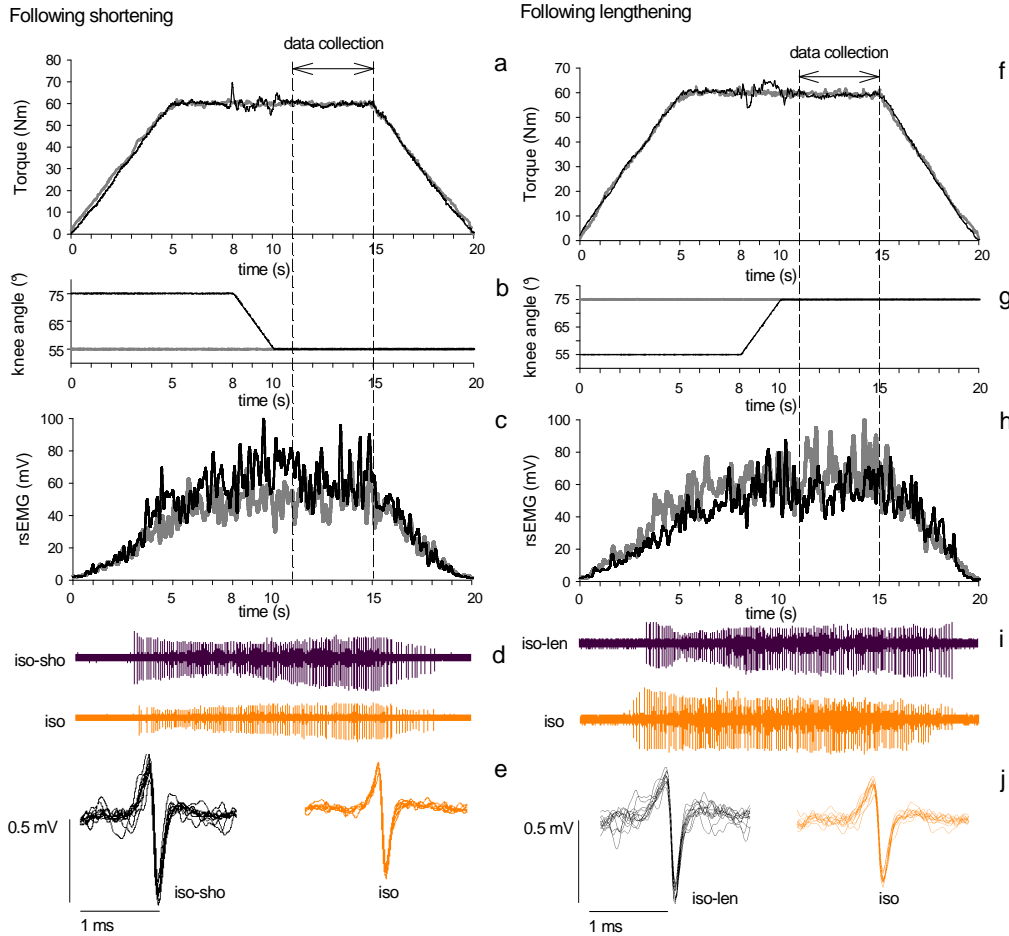


Figure 4.1. Rectified surface EMG (rsEMG) and single motor unit action potentials (SMUAPs) of the same motor unit for one subject during isometric reference contractions (iso, grey traces) and an isometric contraction following shortening (iso-sho, left panel) and lengthening (iso-len, right panel; black traces). In (a and f) the produced torque is shown, in (b and g) the change in knee angle, in (c and h) the rsEMG, in (d and i) the intramuscular EMG, and in (e and j) the consecutive superimposed SMUAPs, each SMUAP is the averaged waveform of 1 s. The comparison of EMG between isometric reference contractions and following shortening or lengthening was made between 11 and 15 s, with the muscles contracting isometrically at the same knee angle. For this figure intramuscular EMG from the same channel is shown to demonstrate the similar shape of the SMUAPs across trials, however, for the motor unit analysis sometimes one of the other channels was used. Note that in this figure the rsEMG was additionally low-pass filtered at 5 Hz.

Since in the first session the rsEMG at the mid VL position was not significantly different from the distal VL position following shortening and lengthening and during isometric reference contractions and motor unit EMG from the second session was sampled at the mid VL position, only the results of rsEMG from the VL mid position will be presented. Furthermore, the behaviour of rsEMG among the two measured hamstring muscles (BF and ST) was similar ($P > 0.05$) during the first session and since during the second session rsEMG was only obtained from the BF, only the rsEMG of the BF will be presented as an indication for co-contraction.

Muscle activation following shortening

Surface EMG

In line with our expectations, absolute rsEMG for the VM, VL and RF muscles were significantly higher following shortening compared to isometric contractions at the same knee angle and absolute isometric torque, for contractions both at 10 and 50 % MVC and during the whole sampling period (1 – 5 s following shortening) in the first session. This result was consistently found for all 10 subjects. Normalized rsEMG (Table 4.1) was not significantly different among muscles and torque levels. Furthermore, normalized rsEMG following shortening tended to decrease ($P = 0.10$) in time during the first session (data not shown). Similar to the first session, during the second session normalized VL rsEMG was significantly higher 1 s following shortening and tended to decrease ($P = 0.07$) during the next 4 s (Figure 4.2A). Furthermore, the enhancement in normalized VL rsEMG was independent of the torque level ($R^2 = 0.14$, $P > 0.05$).

Table 4.1. Normalized (isometric reference contraction = 1.0) rsEMG (mean \pm SD) of the VM, VL and RF muscles 1 s following shortening and lengthening at 10 and 50 % MVC.

	Following shortening		Following lengthening	
	10 % MVC	50 % MVC	10 % MVC	50 % MVC
VM	1.22 \pm 0.16*	1.14 \pm 0.15*	0.80 \pm 0.15#	0.93 \pm 0.07
VL	1.19 \pm 0.14*	1.09 \pm 0.06*	0.84 \pm 0.12#	0.94 \pm 0.10
RF	1.25 \pm 0.56*	1.21 \pm 0.17*	1.23 \pm 0.27*	0.89 \pm 0.06

*Indicates normalized rsEMG significant higher than 1.0, and # indicates normalized rsEMG lower than 1.0 ($P < 0.05$).

Normalized BF rsEMG (% maximum) 1 s following shortening and during isometric contractions for the first and second session are shown in Table 4.2. During the first session at 50 % MVC but not at 10 % MVC, co-contraction was slightly (only 1.1 %) but significantly higher following shortening compared to isometric contractions. During the second session, co-contraction was not significantly different following shortening. For both sessions, co-contraction did not change in time following shortening ($P > 0.05$, data not shown).

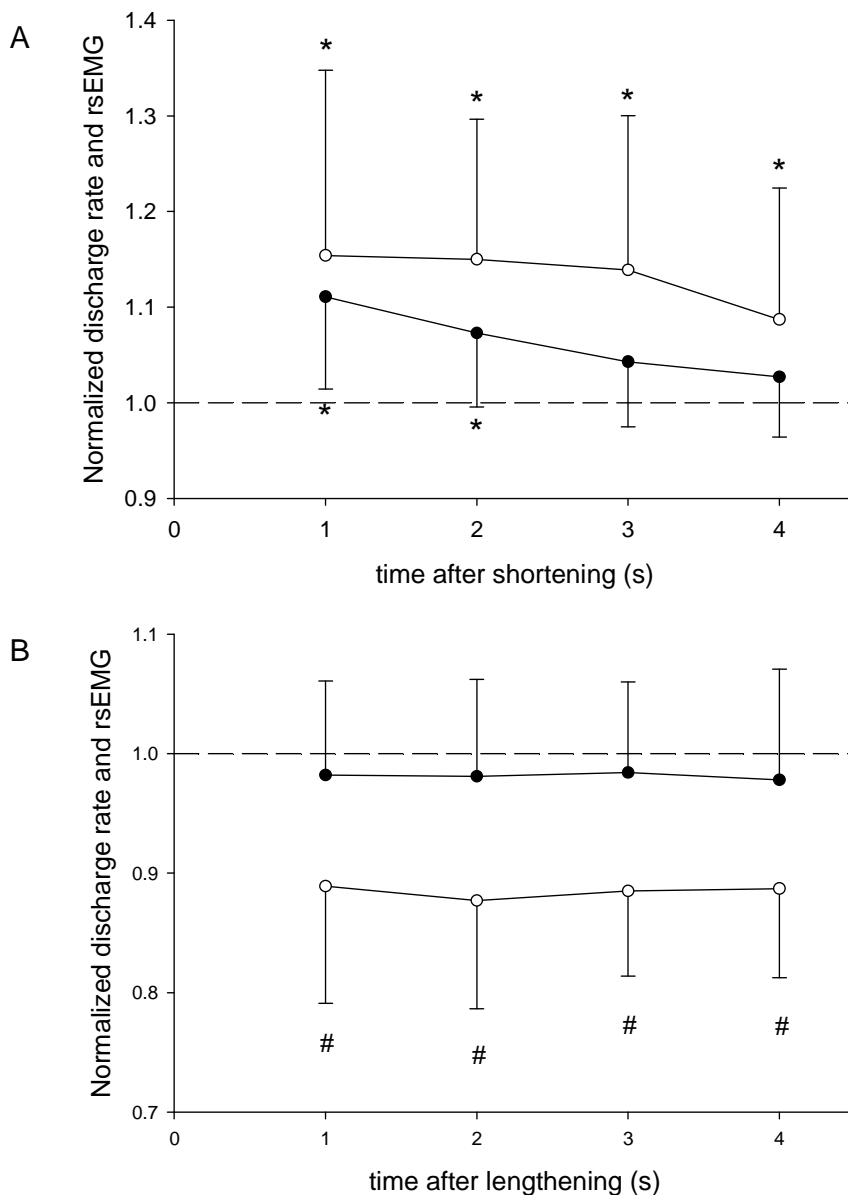


Figure 4.2. Normalized vastus lateralis motor unit firing rates (closed circles) and VL rsEMG (open circles) 1, 2, 3 and 4 s following shortening (A) and following lengthening (B) at on average 18.1 ± 13.3 % and 18.7 ± 13.3 % MVC respectively. * Indicates significant higher normalized firing rate and VL rsEMG following shortening, normalized firing rate significantly decreased and normalized rsEMG tended to decrease ($P = 0.07$) in time following shortening. #Indicates significant lower VL rsEMG following lengthening, normalized firing rate and rsEMG did not change significantly in time.

Motor unit EMG

Forty two motor units were studied following shortening. Relative torque levels varied from 4 up to 47 % MVC and were on average 18.1 ± 13.3 % MVC. Discharge rates at 1, 2, 3 and 4 s following shortening were on average respectively 12.8 ± 1.2 , 12.3 ± 1.3 , 12.0 ± 1.2 and 11.8 ± 1.4 Hz and for the isometric contractions respectively 11.6 ± 1.4 , 11.5 ± 1.5 , 11.5 ± 1.4 and 11.5 ± 1.4 Hz at the same knee angle and absolute

isometric torque. Similar to the rsEMG normalized discharge rate was significantly higher 1 s following shortening, which significantly decreased during the next 4 s (Figure 4.2A). Moreover, normalized discharge rate was not dependent on the torque produced ($R^2 = 0.05$, $P > 0.05$).

Table 4.2. Normalized (% maximum) BF rsEMG (mean \pm SD) 1 s following shortening (iso-sho), 1 s following lengthening (iso-len) and during isometric reference contractions (iso) at 10 and 50 % MVC (first session) and at 18.1 ± 13.3 and 18.7 ± 13.3 % MVC for contractions following shortening and lengthening respectively (second session).

	Following shortening				Following lengthening		
	10%MVC	50%MVC	18%MVC		10%MVC	50%MVC	19%MVC
Iso-sho	2.3 \pm 0.8	8.8 \pm 3.7*	9.5 \pm 4.7	Iso-len	1.7 \pm 0.9	7.8 \pm 4.2	7.9 \pm 4.5
Iso	2.3 \pm 1.6	7.7 \pm 3.1	8.1 \pm 4.8	Iso	2.2 \pm 1.3	8.1 \pm 4.8	9.0 \pm 5.1

*Indicates significant higher normalized BF rsEMG following shortening compared to isometric reference contraction ($P < 0.05$).

Muscle activation following lengthening

Surface EMG

The normalized rsEMG of the RF muscle was significantly different from the rsEMG of the VM and VL muscle at 10 % but not at 50 % MVC in the first session (Table 4.1). Therefore absolute rsEMG for the RF muscle was tested separately from the rsEMG of the VM and VL muscles at 10 % MVC. At 10 % MVC absolute rsEMG for the VM and VL were lower following lengthening compared to isometric contractions at the same knee angle and the same absolute isometric torque ($P < 0.05$). In contrast absolute rsEMG of the RF was higher following lengthening ($P < 0.05$). At 50 % MVC absolute rsEMG for the VM, VL and RF muscles tended to be lower ($P = 0.07$) following lengthening. Although in the majority of contractions rsEMG following lengthening was lower, for all subjects sometimes higher rsEMG values were recorded following lengthening. For the first session, at both torque levels and in all muscles, there were no significant changes in time of the normalized rsEMG following lengthening (data not shown). For the second session, normalized rsEMG of the VL was significantly lower following lengthening and remained constant in time ($P > 0.05$) for the next 4 s (Figure 4.2B). Furthermore, the reduction in VL rsEMG was not dependent on the torque exerted ($R^2 = 0.02$, $P > 0.05$).

Normalized BF rsEMG (% maximum) following lengthening and during isometric contractions for the first and second session are shown in Table 4.2. Co-contraction was similar following lengthening compared to isometric contractions for both the first and the second session and during the whole sampling period ($P > 0.05$).

Motor unit EMG

The discharge behaviour of 40 of the 42 motor units studied following shortening could also be studied following lengthening. Relative torque levels varied from 4 up to 47 % MVC and were on average 18.7 ± 13.3 % MVC. Discharge rates at 1, 2, 3 and 4 s following lengthening were on average respectively 12.1 ± 1.9 , 11.9 ± 1.9 , 11.9 ± 1.9 and 11.7 ± 1.8 Hz, and for the isometric contraction respectively 12.1 ± 1.4 , 12.0 ± 1.4 , 12.0 ± 1.3 and 12.0 ± 1.3 Hz at the same knee angle and at the same absolute isometric torque. Normalized discharge rates following lengthening are shown in Figure 4.2B. In contrast to our expectations, normalized discharge rate was not significantly lower following lengthening and remained constant in time ($P > 0.05$).

Discussion

The purpose of the present study was to establish at the motor unit level whether quadriceps muscle activation was enhanced following shortening and depressed following lengthening compared to isometric reference contractions at the same joint angle and absolute isometric torque. The main findings were that both rsEMG and discharge rate were higher following shortening compared to isometric contractions, which was in line with our expectations. Furthermore, this increase in muscle activation following shortening was not related to the torque (4 – 47 % MVC) exerted. Following lengthening rsEMG was lower compared to isometric reference contractions; however, discharge rate was not different following lengthening.

Following shortening

In line with our hypothesis, the activation of the quadriceps muscle was higher following shortening compared to the isometric reference contraction of the same duration and performed at the same knee angle, indicating that shortening induced force depression was present. Thus the reduced force capacity following shortening was compensated for by an increase in quadriceps muscle activation, and specifically this was accomplished by increasing the discharge rate of already activated motor units. Although a quantitative comparison between changes in discharge rate and rsEMG is not possible the larger increase in rsEMG (~15 %) compared to the increase in discharge rate (~10 %) can partially be accounted for by additional recruitment of motor units following shortening (Figure 4.2A). It may be argued that surface EMG is not a very good measure of muscle activation, particularly during the dynamic phase of the contraction where electrodes may shift relative to the muscle fibres (Farina, 2006;

Farina *et al*, 2001). However, we only made comparisons of surface EMG between isometric (phases of) contractions performed at the same knee angle. Moreover, the consistency of the higher rsEMG following shortening within and among the two sessions in the present study is striking and therefore we assume that the surface EMG findings were a valid measure of the increase in muscle activation following shortening.

Force depression increases linearly with the amount of shortening and decreases with increases in the speed of shortening (de Ruiter and de Haan, 2003; de Ruiter *et al*, 1998; Lee and Herzog, 2003). Additionally, during electrically evoked contractions force depression was found to be lower at lower activation levels and therefore it was suggested that force depression is strongly related to the amount of force or work performed during the shortening phase (de Ruiter *et al*, 1998; Herzog and Leonard, 1997; Herzog *et al*, 1998; Herzog *et al*, 2000; Leonard and Herzog, 2005). However, during the voluntary contractions of the present study the enhancement of muscle activation (both rsEMG and discharge rate) was not increased when higher torques were exerted. In line with our findings, Rousanoglou *et al* (2007) suggested that the relative amount of force depression was not affected by the level of activation (or force) since in two studies of this group a similar amount of force depression was found during submaximal voluntary contractions (~15 %, (Rousanoglou *et al*, 2007)) and following maximal voluntary contractions (~17 %, (Lee and Herzog, 2003)) in the adductor pollicis muscle. In the present study we also conclude that the increase in muscle activation due to shortening induced force depression was not torque dependent, which is not in line with the theory of a stress-dependent inhibition of cross-bridge attachment, predicting that force depression would be higher with greater forces during the contraction (Herzog, 1998; Herzog and Leonard, 2007; Marechal and Plaghki, 1979). It is unclear why the increase in muscle activation following shortening was not dependent on the torque (work) produced. The different way in which motor units are activated during electrically evoked and voluntary submaximal contractions could somehow play a role. During maximal electrically stimulated contraction with submaximal stimulation frequencies, all motor units are activated in a synchronized way (de Ruiter *et al*, 1998; Herzog and Leonard, 1997; Leonard and Herzog, 2005), and with submaximal electrical currents a smaller number of motor units is activated in a synchronized manner (Herzog *et al*, 1998; Herzog *et al*, 2000). However, during voluntary submaximal contractions only the smaller motor units of the knee extensors will be asynchronously activated, especially within the torque range (4 – 47 %) investigated in the present study.

Force depression is long lasting as it could persist for 15 – 30 s following shortening (Herzog *et al*, 1998; Lee and Herzog, 2003) and a steady force is often not reached within ~6 s (de Ruiter and de Haan, 2003; Herzog *et al*, 1998; Lee and Herzog, 2003). Therefore, the magnitude of the force depression, or conversely enhancement of muscle activation, depends on the time of calculation following shortening. To investigate these effects, muscle activation was measured at different times (1 – 5 s) following the shortening contractions. We found that normalized rsEMG tended to decrease in the first ($P = 0.10$) and second session ($P = 0.07$), and normalized motor unit discharge rates significantly decreased with time following shortening (Table 4.1, Figure 4.2A). These findings indicate that during submaximal voluntary contractions the increased muscle activation following shortening declined within a few seconds following shortening. This is in line with previous studies where a decline of force depression over the first seconds following shortening during maximal activation was found (de Ruiter and de Haan, 2003; Herzog *et al*, 1998; Lee and Herzog, 2003).

Following lengthening

Following lengthening contractions at 10 % MVC (first session) activation of the RF muscle increased and decreased for the VM and VL muscles compared to isometric reference contractions, while at 50 % MVC (first session) and at 18.3 ± 13.3 % MVC (second session) the changes among the quadriceps components were similar. These torque dependent differences in rsEMG among the knee extensor muscles could signify a task related adaptation in activation and the ~20 % decrease in rsEMG of the VM and VL following lengthening contractions at 10 % MVC could at least partially be an adjustment to the increased muscle activation to the RF muscle during the same contractions (Table 4.1).

RsEMG was ~10 % lower ($P < 0.05$) in all muscles ($P > 0.05$) following lengthening compared to isometric reference contractions. A 10 % lower rsEMG following lengthening would be in agreement with the ~12 % lower rsEMG following lengthening found during submaximal contractions at 30 % MVC in the adductor pollicis muscle (Oskouei and Herzog, 2005). This 12 % was recalculated from the data of Oskouei & Herzog (2005) when the changes in rsEMG of all subjects (those who showed force enhancement and those who did not) were taken into account. One explanation for the decrease in rsEMG could be that the motor units were activated in a less synchronized manner following lengthening. However, the rsEMG remained decreased 4 s following lengthening, and since the level of synchronization was not

expected to be different from an isometric contraction at the same absolute torque and knee angle at this point in time, the decrease in rsEMG following lengthening was probably not caused by a decreased synchronization of motor units. As motor unit discharge rate in the present study was not changed following lengthening compared to isometric reference contractions, it may be speculated that the ~10 % decrease in rsEMG indicated that the number of activated motor units was decreased in response to the enhanced force capacity of the contracting muscle fibres following lengthening (Figure 4.2B).

In the studies of Oskouei & Herzog (2005; Oskouei and Herzog, 2006a; Oskouei and Herzog, 2006b) force enhancement was increased with increasing levels of activation during the stretch, suggesting that force enhancement could not just be caused by passive (non cross-bridge) structures. However, in contrast to Oskouei & Herzog (2005; Oskouei and Herzog, 2006a; Oskouei and Herzog, 2006b), but in line with de Ruiter *et al* (2000), we did not find a larger decrease in muscle activation (indicative for greater force enhancement) following lengthening at higher torques. This independency of the decrease in quadriceps muscle activation on the torque produced support the suggestion that force enhancement has characteristics that seem unrelated to the cross-bridge properties (de Ruiter *et al*, 2000; Edman and Tsuchiya, 1996; Pinniger *et al*, 2006).

In the present study variations in quadriceps muscle-tendon complex were induced by varying the knee angle. It should be noted that the changes in the muscle-tendon complex length lead to changes in the length of the compliant patellar tendon, resulting in relatively small changes in fibre length. Moreover, since the shortening and lengthening occurred over a small range of knee angles and at a low speed, the variations in knee angle and thus muscle fibre length were small and relatively slow. Due to these small and slow changes, the length of the fibres and the tendon were expected to be constant during the 1 – 5 s following shortening and lengthening. The small changes in quadriceps muscle fibre length further suggest that the changes in muscle activation following shortening and lengthening contractions might be even larger during daily life movements than found in the present study.

To summarize, in line with our expectations we have demonstrated that both rsEMG and discharge rates were enhanced following shortening compared to isometric reference contractions at the same knee angle and when the same absolute isometric torque was produced. Within the torque range studied (4 – 47 % MVC) this enhancement of muscle activation was not related to the torque produced during the contractions. In contrast, following lengthening rsEMG, but not discharge rate, was

decreased compared to isometric reference contractions at the same knee angle when the same absolute isometric torque was produced. We therefore conclude that activation of the quadriceps muscle was enhanced in response to the reduced force capacity following shortening, mainly by increasing the discharge rate of the already activated motor units (rate coding) and probably by the recruitment of additional motor units, whereas quadriceps muscle activation was decreased in response to the enhanced force capacity following lengthening, possibly by derecruitment of motor units.

References

- Abbott BC, Aubert XM (1952). The force exerted by active striated muscle during and after change of length. *J Physiol* 117: 77-86.
- Cook CS, McDonagh MJN (1995). Force responses to controlled stretches of electrically stimulated human muscle-tendon complex. *Exp Physiol* 80: 477-490.
- de Ruiter CJ, de Haan A (2003). Shortening-induced depression of voluntary force in unfatigued and fatigued human adductor pollicis muscle. *J Appl Physiol* 94: 69-74.
- de Ruiter CJ, de Haan A, Jones DA, Sargeant AJ (1998). Shortening-induced force depression in human adductor pollicis muscle. *J Physiol* 507: 583-591.
- de Ruiter CJ, Didden WJ, Jones DA, de Haan A (2000). The force-velocity relationship of human adductor pollicis muscle during stretch and the effects of fatigue. *J Physiol* 526: 671-681.
- de Ruiter CJ, Elzinga MJ, Verdijk PW, van Mechelen W, de Haan A (2005). Changes in force, surface and motor unit EMG during post-exercise development of low frequency fatigue in vastus lateralis muscle. *Eur J Appl Physiol* 94: 659-669.
- de Ruiter CJ, Elzinga MJH, Verdijk PWL, van Mechelen W, de Haan A (2004). Voluntary drive-dependent changes in vastus lateralis motor unit firing rates during a sustained isometric contraction at 50% of maximum knee extension force. *Pflugers Arch - Eur J Physiol* 447: 436-444.
- Edman KAP, Caputo C, Lou F (1993). Depression of tetanic force induced by loaded shortening of frog fibres. *J Physiol* 466: 535-552.
- Edman KAP, Elzinga G, Noble MIM (1982). Residual force enhancement after stretch of contracting frog single muscle fibers. *J Gen Physiol* 80: 769-784.
- Edman KAP, Tsuchiya T (1996). Strain of passive elements during force enhancement by stretch in frog muscle fibres. *J Physiol* 490: 191-205.
- Farina D (2006). Interpretation of the surface electromyogram in dynamic contractions. *Exerc Sport Sci Rev* 34: 121-127.
- Farina D, Merletti R, Nazzaro M, Caruso I (2001). Effect of joint angle on EMG variables in leg and thigh muscles. *IEEE Eng Med Biol Mag* 20: 62-71.
- Granzier HLM, Pollack GH (1989). Effect of active pre-shortening on isometric and isotonic performance of single frog muscle fibres. *J Physiol* 415: 299-327.
- Hahn D, Seiberl W, Schwirtz A (2007). Force enhancement during and following muscle stretch of maximal voluntarily activated human quadriceps femoris. *Eur J Appl Physiol* 100: 701-709.

- Herzog W (1998). History dependence of force production in skeletal muscle: a proposal for mechanisms. *J Electromyogr Kinesiol* 8: 111-117.
- Herzog W, Leonard TR (1997). Depression of cat soleus-forces following isokinetic shortening. *J Biomech* 30: 865-872.
- Herzog W, Leonard TR (2002). Force enhancement following stretching of skeletal muscle: a new mechanism. *J Exp Biol* 205: 1275-1283.
- Herzog W, Leonard TR (2005). The role of passive structures in force enhancement of skeletal muscles following active stretch. *J Biomech* 38: 409-415.
- Herzog W, Leonard TR (2007). Residual force depression is not abolished following a quick shortening step. *J Biomech* 40: 2806-2810.
- Herzog W, Leonard TR, Wu JZ (1998). Force depression following skeletal muscle shortening is long lasting. *J Biomech* 31: 1163-1168.
- Herzog W, Leonard TR, Wu JZ (2000). The relationship between force depression following shortening and mechanical work in skeletal muscle. *J Biomech* 33: 659-668.
- Herzog W, Schachar R, Leonard TR (2003). Characterization of the passive component of force enhancement following active stretching of skeletal muscle. *J Exp Biol* 206: 3635-3643.
- Lee HD, Herzog W (2002). Force enhancement following muscle stretch of electrically stimulated and voluntarily activated human adductor pollicis. *J Physiol* 545: 321-330.
- Lee HD, Herzog W (2003). Force depression following muscle shortening of voluntarily activated and electrically stimulated human adductor pollicis. *J Physiol* 551: 993-1003.
- Lee HD, Suter E, Herzog W (1999). Force depression in human quadriceps femoris following voluntary shortening contractions. *J Appl Physiol* 87: 1651-1655.
- Lee HD, Suter E, Herzog W (2000). Effects of speed and distance of muscle shortening on force depression during voluntary contractions [In Process Citation]. *J Biom* 33: 917-923.
- Leonard TR, Herzog W (2005). Does the speed of shortening affect steady-state force depression in cat soleus muscle? *J Biomech* 38: 2190-2197.
- Marechal G, Plaghki L (1979). The deficit of the isometric tetanic tension redeveloped after a release of frog muscle at a constant velocity. *J Gen Physiol* 73: 453-467.
- Oskouei AE, Herzog W (2005). Observations on force enhancement in submaximal voluntary contractions of human adductor pollicis muscle. *J Appl Physiol* 98: 2087-2095.

- Oskouei AE, Herzog W (2006a). The dependence of force enhancement on activation in human adductor pollicis. *Eur J Appl Physiol* 98: 22-29.
- Oskouei AE, Herzog W (2006b). Force enhancement at different levels of voluntary contraction in human adductor pollicis. *Eur J Appl Physiol* 97: 280-287.
- Pinniger GJ, Cresswell AG (2007). Residual force enhancement after lengthening is present during submaximal plantar flexion and dorsiflexion actions in humans. *J Appl Physiol* 102: 18-25.
- Pinniger GJ, Ranatunga KW, Offer GW (2006). Crossbridge and non-crossbridge contributions to tension in lengthening rat muscle: force-induced reversal of the power stroke. *J Physiol* 573: 627-643.
- Rousanoglou EN, Oskouei AE, Herzog W (2007). Force depression following muscle shortening in sub-maximal voluntary contractions of human adductor pollicis. *J Biomech* 40: 1-8.

